REMARKS

With the cancellation of claims 1-147, 152-174 and 176, and the addition of claims 177-191, claims 148-151, 175, and 177-191 are pending. The new claims 177 and 178 are supported by the specification at page 22, lines 1-2. The new claims 179-189 are supported by the specification at page 2, line 30 to page 3, line 1 and page 18, lines 1-18. The new claims 177-191 are directed toward methods of treating an autoimmune disease comprising administering a composition containing at least one boswellic acid compound, so claims 177-191 fall within the elected group.

Applicants submit that the amendments to claims 148 and 175 fail to narrow the scope of the amended claim recitation because the subject being treated in the method of claim 148 or 175 is, despite the amendment, still the same.

Claim Rejection -- 35 U.S.C. 112, First Paragraph

Claims 144-147, 162 and 174 were rejected as non-enabled because of the prevention of an autoimmune disease. Applicants respectfully traverse the rejection. Without acquiescence with the rejection, claims drawn toward methods of preventing the autoimmune disease have been canceled. Withdrawal of the rejection is requested.

Claim Rejection -- 35 U.S.C. 103

(A) Claims 144-151, 162, 174 and 175 were rejected as obvious over Nagasawa (the English abstract of JP 04288095) in view of Shao et al ("Inhibitory activity of boswellic acids against human leukemia HL-60 cells in culture", Planta Medica, vol. 64, pp. 328-331, 1998). Applicants respectfully traverse the rejection.

The Examiner alleged that Nagasawa discloses that beta-boswellic acids such as beta-boswellic acid, acetyl-beta-boswellic acid, 11-keto-beta-boswellic acid and acetyl-11-keto-beta-boswellic acid are useful in treating autoimmune diseases. Applicants respectfully disagree.

The Examiner relied upon the English abstract of Nagasawa as the primary reference. According to the English abstract, the purpose of Nagasawa was to provide a complement activity-inhibiting agent containing **a specific** β-boswellic acid **derivative** as **an** active ingredient, wherein the agent is effective for treating autoimmune diseases (see Purpose of the English abstract; emphasis added). However, the English abstract does not specifically state which compound or salt is the **specific** β-boswellic acid **derivative** used as the active ingredient of the agent. The Constitution portion of the English abstract of Nagasawa discloses that β-boswellic acid acetate was **hydrolyzed** to provide the objective compound (salt) of the formula shown in the Abstract. Because the authors of Nagasawa prepared the β-boswellic acid derivative by hydrolysis of β-boswellic acid acetate, Nagasawa could not have disclosed that acetyl-β-boswellic acid or acetyl-11-keto-β-boswellic acid is effective in treating

autoimmune diseases (because the acetyl group of β-boswellic acid acetate was removed by hydrolysis so the compound or salt tested by the authors of Nagasawa could not have contain the acetyl group). In contrast, the methods of the pending claims comprise the administration of a composition containing at least acetyl-β-boswellic acid or acetyl-11-keto-β-boswellic acid.

Furthermore, some of the pending claims differ from Nagasawa by administering a composition containing β-boswellic acid. But Nagasawa concentrated on the **derivative** of β-boswellic acid. Thus, the authors of Nagasawa could not have disclosed the effect of β-boswellic acid on autoimmune diseases.

Applicants note that Shao et al fails to remedy the deficiencies of Nagasawa because Shao et al does not disclose that a composition containing acetyl-11-keto-β-boswellic acid, acetyl-β-boswellic acid or β-boswellic acid is effective in treating autoimmune diseases. The Examiner stated that Shao et al discloses that the IC50s of 4 boswellic acid compounds (acetyl-11-keto-β-boswellic acid, acetyl-β-boswellic acid, 11-keto-β-boswellic acid and β-boswellic acid) for inhibiting human leukemia HL-60 cells. Applicants contend, however, that the disclosure of Shao et al on the effect of the 4 boswellic acid compounds in inhibiting the growth of HL-60 cells is not relevant to the treatment of autoimmune diseases. After all, the HL-60 cells are cancer cells and any inhibitory effect on the growth of these cancer cells shown by Shao et al does not necessarily have anything to do with autoimmune diseases and is not reasonably predictive of the effect of these boswellic acid compounds on the treatment of

autoimmune diseases. Thus, a person of ordinary skill in the art would not have been motivated to use the teaching of Shao et al to modify the method of Nagasawa by administering acetyl-11-keto-β-boswellic acid in the method of Nagasawa for treating an autoimmune disease. Similarly, a person of ordinary skill in the art would not have been motivated to use the teaching of Shao et al to modify the method of Nagasawa by adding acetyl-β-boswellic acid in the method of Nagasawa. By the same token, a person of ordinary skill in the art would not have been motivated to use the teaching of Shao et al to modify the method of Nagasawa by adding β-boswellic acid in the method of Nagasawa.

In addition, the disclosure by Nagasawa of the inhibitory effect of the β-boswellic acid derivative on complement activity is not reasonably predictive of the effect of the β-boswellic acid derivative on the treatment of autoimmune diseases. This is because the exact cause of autoimmune diseases is not known and the pathology of autoimmune disease is very complex, so a person of ordinary skill in the art would not have reasonably expected that a β-boswellic acid derivative having inhibitory effects of the complement would succeed in treating an autoimmune disease.

Withdrawal of the rejection is requested.

(B) Claims 144-151, 162, 174 and 175 were rejected as obvious over Taneja (EP 0755940). Applicants respectfully traverse the rejection.

The Examiner rejected the claims because Taneja discloses that the 4 boswellic acid compounds (acetyl-11-keto-β-boswellic acid, acetyl-β-boswellic

acid, 11-keto-β-boswellic acid and β-boswellic acid) are effective in treating inflammation and because the Examiner asserted that "inflammation" is broad enough to encompass "autoimmune diseases". Applicants respectfully disagree.

Inflammation and autoimmune diseases are two different health disorders. "Inflammation" is a pathologic process consisting of a dynamic complex of cellular and chemical reactions in response to an injury or abnormal stimulation caused by a physical, chemical, or biologic agent (please see definition from Stedman's Medical Dictionary, 2000, attached). "Autoimmune diseases" are diseases caused by cells or antibodies arising from and directed against an individual's own tissues (please see definition of "autoimmune" from Stedman's Medical Dictionary). A substance effective in treating inflammation is not necessarily effective in treating autoimmune diseases. For example, aspirin is anti-inflammatory, but not effective in treating autoimmune diseases. Thus, the instant claims should not have been rejected as obvious over Taneja.

Conclusion

In view of the above, applicants submit that the application is in a condition for allowance.

In the event that this paper is deemed not timely, applicants petition for an appropriate extension of time. The Commissioner is hereby authorized to charge any fee deficiency or credit any overpayment associated with this communication to Deposit Account No. 01-2300 referencing the docket number of 108064-00049.

Respectfully submitted,

King L. Wong

King L. Wong
Registration No. 37,500

Customer No. 004372
ARENT FOX KINTNER PLOTKIN & KAHN, PLLC
1050 Connecticut Avenue, N.W.,
Suite 400
Washington, D.C. 20036-5339
Tel: (202) 857-6000
Fax: (202) 638-4810

Enclosure: Pages 172, 897 and 898, Stedman's Medical Dictionary, 2000

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